U.S. Department of Labor

Office of Administrative Law Judges
800 K Street, NW, Suite 400-N
Washington, DC 20001-8002

(202) 693-7300
(202) 693-7365 (FAX)

Issue Date: 29 November 2006

In the Matter of

L.A.,
Claimant,

v.

FOUNTAIN BLUE COAL COMPANY, INC.,
Employer,

and

LIBERTY MUTUAL INSURANCE COMPANY,

Carrier,

and

DIRECTOR, OFFICE OF WORKERS' COMPENSATION PROGRAMS,
Party-In-Interest.

Case No. 2004-BLA-00148

Appearances: Joseph E. Wolfe, Esq. For the Claimant
Francesca Maggard, Esq. For the Employer

Before: William S. Colwell
Administrative Law Judge

DECISION AND ORDER DENYING BENEFITS

This proceeding arises from a claim for benefits under the Black Lung Benefits Act, 30 U.S.C. § 901 *et seq*. The Act and applicable implementing regulations, 20 CFR Parts 718 and 725, provide compensation and other benefits to living coal miners who are totally disabled due to pneumoconiosis and their dependents, and surviving dependents of coal miners whose death was due to pneumoconiosis. The Act and regulations define pneumoconiosis, commonly known as black lung disease, as a chronic dust disease of the lungs and its sequelae, including respiratory and pulmonary

impairments, arising out of coal mine employment. 30 U.S.C. § 902(b); 20 CFR § 718.201 (2004). In this case, the Claimant alleges that her husband was a miner, he suffered from pneumoconiosis, and pneumoconiosis hastened his death.

I conducted a hearing on this claim on June 29, 2005, in Pikeville, KY. All parties were afforded a full opportunity to present evidence and argument, as provided in the Rules of Practice and Procedure before the Office of Administrative Law Judges, 29 CFR Part 18 (2004). At the hearing, Director's Exhibits ("DX") 1-53 and Claimant's Exhibit ("CX") 1 were admitted into evidence. Transcript ("Tr.") at 11, 15. Although Claimant objected to DX 37, I shall consider Claimant's objections in determining its evidentiary weight. See post-hearing Order dated September 28, 2005. A post-hearing conference call was held on September 27, 2005. I admitted Employer's Exhibit ("EX") 1 without objection. *Id*. The record was held open after the hearing to allow the parties to submit additional argument. The Claimant and Employer submitted closing arguments, and the record is now closed.

In reaching my decision, I have reviewed and considered the entire record pertaining to the claim before me, including all exhibits admitted into evidence, the testimony at hearing, and the arguments of the parties.

PROCEDURAL HISTORY

The Claimant filed her original claim for survivor's benefits under the Act on December 1, 1997. DX 1. On September 2, 1998, a Claims Examiner informed the Claimant that she was eligible for benefits. DX 44, p. 6. On September 4, 1998, the District Director of the Office of Workers' Compensation Programs ("OWCP") affirmed the initial finding of eligibility. DX 44, p. 2. Fountain Blue Coal Company, Inc., ("Employer") timely appealed that determination. DX 47. The claim was referred to the Office of Administrative Law Judges on March 26, 1999. DX 49, p. 4.

Administrative Law Judge ("ALJ") Thomas F. Phalen conducted a formal hearing on September 16, 1999, in Prestonburg, KY. DX 51, p. 186. ALJ Phalen issued a Decision and Order on November 19, 1999, awarding survivor's benefits. DX 51, pp. 164-180. He determined that the miner had at least 22 years of coal mine employment, that he suffered from pneumoconiosis pursuant to § 718.202(a)(4), and that his death was due to pneumoconiosis. *Id*. Employer filed a notice of appeal on December 14, 1999. DX 51, p. 161. On January 31, 2001, the Benefits Review Board ("BRB") issued a Decision and Order vacating ALJ Phalen's findings that the miner had pneumoconiosis and that his death was caused by pneumoconiosis. DX 51, pp. 103-105. The case was remanded to ALJ Phalen. DX 51, p. 105.

ALJ Phalen issued a Decision and Order on Remand Denying Benefits dated August 31, 2001. DX 51, p. 56. ALJ Phalen determined that the Claimant did not establish that her husband suffered from pneumoconiosis or that his death was caused by pneumoconiosis. DX 51, pp. 7-9. On September 28, 2001, the Claimant appealed to the BRB. The BRB determined that ALJ Phalen's first Decision and Order Awarding

Benefits was null and returned the parties to the *status quo* before the initial decision. DX 51, p. 21. The BRB then affirmed the denial of benefits in a Decision and Order dated July 30, 2002. DX 51, p. 18.

On July 25, 2003, the Claimant filed a request for modification. DX 51, p. 12. The claim was referred to the Office of Administrative Law Judges on August 2, 2004. DX 52. The undersigned ALJ conducted a formal hearing on this matter on June 29, 2005, in Pikeville, KY.

APPLICABLE STANDARDS

This claim was originally filed on December 1, 1997. DX 1. The current regulations at 20 CFR Parts 718 and 725 do not apply; this case will be adjudicated under the regulations enacted before January 1, 2001. Thus, the amount of medical evidence to be considered is not limited by the regulations. Entitlement to benefits must be established under the regulatory criteria at Part 718. *See Neeley v. Director, OWCP*, 11 B.L.R. 1-85 (1988). The Act provides that benefits are provided to eligible survivors of a miner whose death was due to pneumoconiosis. § 718.205(a). In order to receive benefits, the Claimant must prove that:

1. The miner had pneumoconiosis;

2. The miner's pneumoconiosis arose out of coal mine employment; and

3. The miner's death was due to pneumoconiosis.

§§ 718.205(a). Failure to establish any of these elements by a preponderance of the evidence precludes entitlement. *See Anderson v. Valley Camp of Utah, Inc.*, 12 B.L.R. 1-111, 1-112 (1989); *Trent v. Director, OWCP*, 11 B.L.R. 1-26, 1-27 (1987). The Board has held that, in a survivor's claim under Part 718, the ALJ must make a threshold determination as to the existence of pneumoconiosis under 20 C.F.R. § 718.202(a) (2000) prior to considering whether the miner's death was due to pneumoconiosis. *Trumbo v. Reading Anthracite Co.*, 17 B.L.R. 1-85 (1993).

ISSUES

After the hearing, the following are the remaining contested issues:

1. Whether the miner had pneumoconiosis as defined by the Act and the regulations.

2. Whether the miner's pneumoconiosis arose out of his coal mine employment.

3. Whether the miner's death was due to pneumoconiosis.

4. Whether the evidence establishes a change in conditions and/or that a mistake was made in the determination of any fact in the prior denial per 20 C.F.R. § 725.310.

DX 52, pp. 1-2; Tr. 6-7. (The Employer withdrew the issues of miner and Responsible Operator at the hearing. The Employer also stipulated to 22 years of coal mine employment.)

The Employer also reserved its right to challenge the statute and regulations. These issues are beyond the authority of the administrative law judge and are preserved for appeal purposes only. DX 52, p. 2.

FINDINGS OF FACT AND CONCLUSIONS OF LAW

Factual Background and the Claimant's Testimony

The Claimant did not testify at the most recent hearing on June 29, 2005. The parties stipulated that she has not remarried since the miner's death and that she is not receiving Kentucky Workers' Compensation benefits. Tr. 17-18. The Claimant testified at the prior hearing on September 16, 1999. Her testimony and other relevant background information is summarized in ALJ Phalen's prior Decisions and Orders and are incorporated by reference, as if set forth fully herein.

The miner died on October 26, 1997. DX 11. The causes of death were listed as respiratory failure and idiopathic pulmonary fibrosis. DX 11.

The miner's last coal mine employment was in Kentucky. DX 20. Therefore this claim is governed by the law of the 6th Circuit. *Shupe v. Director, OWCP*, 12 B.L.R. 1-200, 1-202 (1989) (*en banc*).

Modification

Section 22 of the Longshore and Harbor Workers' Compensation Act, 33 U.S.C. § 922, as incorporated into the Black Lung Benefits Act by 30 U.S.C. § 932(a) and as implemented by § 725.310, provides that upon the Claimant's own initiative, or upon the request of any party on the ground of a change in conditions or because of a mistake in a determination of fact, the fact-finder may, at any time prior to one year after the date of the last payment of benefits, or at any time before one year after the denial of a claim, reconsider the terms of an award or a denial of benefits. § 725.310(a).

In deciding whether a mistake in fact has occurred, the United States Supreme Court stated that the ALJ has "broad discretion to correct mistakes of fact, whether demonstrated by wholly new evidence, cumulative evidence, or merely further reflection on the evidence initially submitted." *O'Keefe v. Aerojet-General Shipyards, Inc.*, 404 U.S. 254, 256 (1971). Evidence in the entire claim file must be considered to determine whether a "mistake in a determination of fact" was made. This is required even where

no specific mistake of fact has been alleged. *Coal Co. v. Director, OWCP [Worrell]*, 27 F.3d 227 (6th Cir. 1994)*; Jessee v. Director, OWCP*, 5 F.3d 723 (4th Cir. 1993)*; Kingery v. Hunt Branch Coal Co.*, 19 B.L.R. 1-6 (1994)*; Kovac v. BCNR Mining Corp.*, 14 B.L.R. 1-156 (1990), *aff'd on recon.,* 16 B.L.R. 1-71 (1992). In a survivor's claim, the sole ground for modification is that there has been a mistake in a determination of fact. This is because there can be no change in the deceased miner's condition.

This claim was filed in 1997; therefore, it must be adjudicated under the regulations at 20 C.F.R. Part 718 (2000). Accordingly, I will consider all of the prior evidence and the newly submitted evidence: a report by Dr. Joshua Perper dated June 5, 2005, (CX 1) and report by Dr. Peter Tuteur dated August 15, 2005 (EX 1). I will make a *de novo* review of the record to determine whether there has been a mistake in determination of fact.

Medical Evidence

Chest X-rays

Chest x-rays may reveal opacities in the lungs caused by pneumoconiosis and other diseases. Larger and more numerous opacities result in greater lung impairment. The quality standards for chest x-rays and their interpretations are found at 20 C.F.R. § 718.102 (2000) and Appendix A of Part 718. The following table summarizes the x-ray findings available in this case. The existence of pneumoconiosis may be established by chest x-rays classified as Category 1, 2, 3, A, B, or C according to ILO-U/C International Classification of Radiographs. Small opacities (1, 2, or 3) (in ascending order of profusion) may be classified as round (p, q, r) or irregular (s, t, u), and may be evidence of "simple pneumoconiosis." Large opacities (greater than 1 cm) may be classified as A, B or C, in ascending order of size, and may be evidence of "complicated pneumoconiosis." A chest x-ray classified as category "0," including subcategories 0/-, 0/0, 0/1, does not constitute evidence of pneumoconiosis. 20 C.F.R. § 718.102(b) (2000).

Physicians' qualifications appear after their names. Qualifications have been obtained where shown in the record by curriculum vitae or other representations, or if not in the record, by judicial notice of the lists of readers issued by the National Institute of Occupational Safety and Health (NIOSH).[1] If no qualifications are noted for any of the following physicians, it means that I have been unable to ascertain them either from the record or the NIOSH list. Qualifications of physicians are abbreviated as follows: A=

[1]NIOSH is the federal government agency that certifies physicians for their knowledge of diagnosing pneumoconiosis by means of chest x-rays. Physicians are designated as "A" readers after completing a course in the interpretation of x-rays for pneumoconiosis. Physicians are designated as "B" readers after they have demonstrated expertise in interpreting x-rays for the existence of pneumoconiosis by passing an examination. Historical information about physician qualifications appears on the U.S. Department of Health and Human Services, List of NIOSH Approved B-Readers with Inclusive Dates of Approval [as of] June 7, 2004, found at http://www.oalj.dol.gov/public/blalung/refrnc/bread3_07_04.htm. Current information about physician qualifications appears on the CDC/NIOSH, NIOSH Certified B-Readers List found at http://www2a.cdc.gov/drds/breaders/breaders_results.asp.

NIOSH certified A-reader; B= NIOSH certified B-reader; BCR= Board-certified in radiology. Readers who are Board-certified radiologists and/or B-readers are classified as the most qualified. See *Mullins Coal Co. v. Director, OWCP*, 484 U.S. 135, 145 n. 16 (1987); *Old Ben Coal Co. v. Battram*, 7 F.3d 1273, 1276 n.2 (7th Cir. 1993). B-readers need not be radiologists.

Date of X-ray/ reading	Readers' Qualifications	Reading and Film Quality	Result Concerning Presence of Pneumoconiosis
DX 18 02/14/1977 03/01/1998	Dr. E. Nicholas Sargent BCR, B	U/R	Copied film is unacceptable because over-exposed, poor resolution, and mottling.
DX 35, p. 6 02/14/1977 06/11/1998	Dr. Bruce C. Broudy	Negative U/R	There is definitely some post-inflammatory change in the bases with some thickening of the minor fissure, but the films are not of sufficient quality to be interpreted for pneumoconiosis.
DX 41, p. 8 02/14/1977 06/26/1998	Dr. Thomas M. Jarboe B	U/R, upper and mid zones dark. Copy.	
DX 13, p. 171 02/18/1977 02/18/1977	Dr. G.N. Combs		"The heart and great vessels are normal in appearance. There is infiltrate in the posterior basilar segment of the lower left lobe. The remaining lung parenchyma is clear. Bony thorax and diaphragm shadows are normal in appearance. Infiltrate as described, felt to represent pneumonia."

Date of X-ray/ reading	Readers' Qualifications	Reading and Film Quality	Result Concerning Presence of Pneumoconiosis
DX 13, p. 172 02/23/1977 02/23/1977	Dr. G.N. Combs		"Repeat chest film still shows the minimal infiltrate at the left base described on examination of 2/18/1977. There is also a very small area at the right base. Actually, these may represent small areas of discold atelectasis rather than pneumoic infiltrate."
DX 13, p. 157 09/30/1977 09/30/1977	Dr. G.N. Combs		"Minimal infiltrate at both bases having the appearance of discord atelectasis. Compared with previous films on this patient dating back to 2/14/77, there is no significant change in radiographic appearance."
DX 13, p. 158 10/05/1977 10/05/1977	Dr. B. Brandon		"Review of chest radiographs dating back to February, 1977, show persistent and unchanged areas of apparent fibrotic change in both bases." Could not rule out pulmonary embolism.

Date of X-ray/ reading	Readers' Qualifications	Reading and Film Quality	Result Concerning Presence of Pneumoconiosis
DX 13. p. 161 11/04/1977 11/04/1977	Dr. B. Brandon		"The appearance of the chest is stable since prior examinations dating back to February. The multiple linear densities are unchanged. On the basis of this, they are felt to represent the fibrotic residual of prior disease."
DX 13, p. 109 10/06/1982 10/06/1982	Dr. K.B. Kim		"No active cardiopulmonary disease. Discoid atelectasis at the both bases." [*sic*]
DX 13. p. 106 09/17/1983 09/17/1983	Dr. Alex Poulos		"Cardiomegaly. Chronic linear changes at the right lung base. Chronic pleural thickening noted along the lateral chest wall of the right upper thorax. No acute infiltrates are seen in either lung field."
DX 17 09/17/1983 03/01/1998	Dr. E. Nicholas Sargent BCR, B	U/R	Need original film. Copied film unacceptable because over-exposed, poor resolution, and mottling.

Date of X-ray/ reading	Readers' Qualifications	Reading and Film Quality	Result Concerning Presence of Pneumoconiosis
DX 35, p. 11 09/17/1983 06/11/1998	Dr. Bruce C. Broudy	Negative 2, slight underpenetration	Bilateral lower lobe inhomogeneous infiltrates suggesting post-inflammatory change are present. No small rounded opacities, therefore categorized as negative or Category 0 according to the ILO classification system for pneumoconiosis. No evidence of any large opacities or pleural disease. DX 35, p. 4.
DX 41, p. 9 09/17/1983 06/26/1998	Dr. Thomas M. Jarboe B	Negative. Category 0. 1	Other abnormalities. Linear scarring right mid and lower zones and left lower zone adjacent to pleura.
DX 13, p. 101 01/04/1984 01/04/1984	Dr. Alex Poulos		"Chronic fibrotic changes noted in the right lower lung field. No active cardiopulmonary disease noted. No evidence of abnormalities to the left ribs."
DX 48, p. 129 04/22/1987 04/22/1987	Dr. Dennis H. Halbert		Linear areas of scarring versus atelectasis seen probably in the region of the right middle lobe.

Date of X-ray/ reading	Readers' Qualifications	Reading and Film Quality	Result Concerning Presence of Pneumoconiosis
DX 48, p. 101 07/15/1991 07/15/1991	Dr. D. H. Halbert		Appears to be some chronic infiltrates in the right lung base. No definite acute infiltrates are seen. Compared to study of 01/04/1984, there has been no significant change. Chronic changes seen in the right base. No evidence of active disease.
DX 20 07/15/1991 03/01/1998	Dr. E. Nicholas Sargent BCR, B	U/R	Copied film is unacceptable because under-exposed, poor contrast, poor resolution, and mottling.
DX 35, p. 9 07/15/1991 06/11/1998	Dr. Bruce C. Broudy	Negative 3, underpenetration laterally	There are post-inflammatory changes in all zones, but to a lesser extent in the left upper zone. The opacities are coarse, irregular, and too large to be small irregular opacities. There are no small rounded opacities. Categorized as negative of Category 0 according to the ILO classification system for small opacities. No large opacities or pleural disease. DX 35, p. 4.

Date of X-ray/ reading	Readers' Qualifications	Reading and Film Quality	Result Concerning Presence of Pneumoconiosis
DX 41, p. 10 07/15/1991 06/26/1998	Dr. Thomas M. Jarboe B	Negative. Category 0/0. 1	Other abnormalities. Lacy reticular pattern in right upper zone with question of honeycombing medial apex. Fibrotic stranding left lower and mid zones.
DX 14, p. 10 06/12/1992 06/12/1992	Dr. Matt Vuskovich B	Negative 1	Negative. Dr. Vuskovich remarked "diffuse interstitial fibrosis lower zones" in the "Other Comments" section.
DX 48, p. 98 03/17/1993 03/17/1993	Dr. A. Poulos		Chronic interstitial disease is seen throughout both lung fields. No evidence of active infiltrates or pleural abnormalities.
DX 48, p. 93 11/09/1993 11/09/1993	Dr. D. H. Halbert		Chronic changes present in both lungs. No acute infiltrates are seen. No evidence of active disease.
DX 19 11/09/1993 03/01/1998	Dr. E. Nicholas Sargent BCR, B	U/R	Copied film is unacceptable because under-exposed, poor contrast, poor resolution, mottling, and poor inspiration.

Date of X-ray/ reading	Readers' Qualifications	Reading and Film Quality	Result Concerning Presence of Pneumoconiosis
DX 35, p. 10 11/09/1993 06/11/1998	Dr. Bruce C. Broudy	Negative 3, underpenetration laterally	Film appears to be a copy and graded as #3 for diagnostic quality. Categorized as negative for Category 0 for small rounded opacities according to the ILO classification system for pneumoconiosis. The opacities present are coarse, irregular, and too large to be considered small irregular or nodular opacities associated with coal workers' pneumoconiosis. No large opacities or pleural disease is noted. DX 35, p. 3.
DX 41, p. 11 11/09/1993 06/28/1998	Dr. Thomas M. Jarboe B	Negative. Category 0/0. 1	Other abnormalities. Reticulonodular scarring in the right upper zone. Fibrosis right mid/lower zones and left lower zone.
DX 48, p. 87 05/02/1994 05/02/1994	Dr. D. H. Halbert		Infiltrate is seen in the right middle lobe and in the left base.
DX 13, p. 25; DX 48, pp. 86, 82 05/05/1994 05/05/1994	Dr. A. Poulos		Diffuse chronic interstitial disease is seen in both lungs particularly in the lower lung zones. No active cardiopulmonary disease is evident.

Date of X-ray/ reading	Readers' Qualifications	Reading and Film Quality	Result Concerning Presence of Pneumoconiosis
DX 13, p. 26 05/10/1994 05/10/1994	Dr. A. Poulos		Not for diagnosis of pneumoconiosis. Chronic interstitial disease is again seen throughout both lung fields particularly in the lower lung zones. No evidence of active areas of infiltration, regions of volume loss, or acute pleural abnormalities. "Diffuse chronic interstitial pulmonary disease. No active cardiopulmonary disease is noted."
DX 16 05/10/1994[2] 04/20/1998	Dr. Raghu R. Sundaram	1	Idiopathic pulmonary fibrosis.[3]
DX 13, p. 47; DX 48, p. 76 11/24/1995 11/24/1995	Dr. A. Poulos		Diffuse chronic interstitial disease seen throughout both lung fields. There are no acute areas of infiltration or pleural abnormalities identified. No active cardiopulmonary disease is noted.
DX 48, p. 74 07/18/1996 07/18/1996	Dr. Alex Poulos		Chronic diffuse interstitial disease in both lungs. No evidence of active disease.
DX 13, p. 38 01/31/1997 01/31/1997	Dr. D. H. Halbert		"Infiltrates in both lungs. This appears to represent progression of chronic disease."

[2] Dr. Sundaram reviewed two chest x-rays on the same form. DX 16.
[3] There are no markings in Sections 2A through 4A.

Date of X-ray/ reading	Readers' Qualifications	Reading and Film Quality	Result Concerning Presence of Pneumoconiosis
DX 22 01/31/1997 03/01/1998	Dr. E. Nicholas Sargent BCR, B	U/R	Copied film is unacceptable because under-exposed, poor contrast, poor resolution, and poor inspiration.
DX 35, p. 9 01/31/1997 06/11/1998	Dr. Bruce C. Broudy	Negative 3, underpenetration laterally	There are extensive bilateral infiltrates in both lungs. There are coarse, irregular markings with only a few small rounded opacities. The underpenetration makes it difficult to evaluate except only for small areas of the lung which would appear negative for pneumoconiosis. DX 35, p. 3.
DX 41, p. 12 01/31/1997 06/26/1998	Dr. Thomas M. Jarboe B	Negative. Category 0/0. 1	Other abnormalities. Thick fibrosis in the right upper zone. Blotchy patches fibrosis in right mid and lower zones. Also dense fibrosis in the left lower zone.
DX 13, p. 7; DX 48, p. 66 09/06/1997[4] 09/06/1997	Dr. A. Poulos		Diffuse chronic interstitial lung disease noted in both lungs. No active areas of infiltration or pleural disease.

[4] Note: Dr. T. Shawn Caudill discusses this film in his consultative report dated September 9, 1997. DX 48, p. 60. "Markedly shortened inspiration with diffuse nodular densities, right lung greater than left. Density size ranges from 5mm – 1 cm throughout. No effusions or obvious infiltrates. Interstitial fibrotic changes also noted, again right greater than left, upper lung fields more densely scarred than lower lung fields. Red by Radiology as consistent with silicosis." I can find no separate chest x-ray report by Dr. Caudill in the record.

Date of X-ray/ reading	Readers' Qualifications	Reading and Film Quality	Result Concerning Presence of Pneumoconiosis
DX 21 09/06/1997 03/01/1998	Dr. E. Nicholas Sargent BCR, B	U/R	Copied film is unacceptable because under-exposed, poor contrast, poor resolution, and poor inspiration.
DX 35, p. 8 09/06/1997 06/11/1998	Dr. Bruce C. Broudy	Negative U/R underpenetration	Film is poor diagnostic quality because of underpenetration in areas along the lateral chest wall and overpenetration over the left upper and mid zones. Appears to be a copy. There are extensive bilateral infiltrates but no characteristic small rounded opacities of coal workers' pneumoconiosis. However, the film is not of sufficient quality to be interpreted for the presence or absence of pneumoconiosis. DX 35, p. 3.
DX 41, p. 13 09/06/1997 06/26/1998	Dr. Thomas M. Jarboe B	Negative. Category 0/0. 3	Other abnormalities. Extensive fibrosis in the right lung—now destroyed with marked honeycombing. Fibrosis persists at the left base.
DX 16 09/24/1997[5] 04/20/1998	Dr. Raghu R. Sundaram	1	Bilateral honeycombing lung/medialstinal adenopathy hilar adenopathy.[6]

[5] Dr. Sundaram reviewed two chest x-rays on the same form. DX 16.
[6] There are no markings in Sections 2A through 4A.

Date of X-ray/ reading	Readers' Qualifications	Reading and Film Quality	Result Concerning Presence of Pneumoconiosis
DX 48, p. 24 09/24/1997 09/24/1997	Dr. James L. Buck		Bilateral honeycombing of the lungs greater in the periphery and worse in the right lung than left. Mild bronchiectasis. No effusions. Several small nodes seen in the anterior mediastinal soft tissues. Scattered smaller nodes throughout the mediastinum. Scatter large mediastinal nodes. Adenopathy of the pervascular, right hilar, left hilar, and subcarinal nodes. May represent sarcoidosis.
DX 48, p. 23 09/25/1997 09/25/1997	Dr. Joseph G. King		Lungs are poorly inflated. Extensive patchy opacities are noted in both lungs. This appears to represent mixed interstitial and alveolar disease. Differential considerations are extensive and include such entities as edema, pneumonia, UIP, and metastatic disease. Right hilar/right paratracheal mass is noted.

Date of X-ray/ reading	Readers' Qualifications	Reading and Film Quality	Result Concerning Presence of Pneumoconiosis
DX 15, p. 72 10/09/1997 10/09/1997	Dr. Joseph G. King		Pneumothorax again noted on the left and appears small at the lung apex. The lungs are poorly inflated. The peripheral consolidation in the right lung appears slightly improved.
DX 15, p. 73 10/09/1997 10/09/1997	Dr. Joseph G. King		Increase in ill-defined consolidation particularly peripherally in the right lung. This may represent pneumonia or hemorrhage. Pneumothorax is noted on the left and appears small at the lung apex. Diffuse pulmonary parenchymal honeycombing is again noted.
DX 15, p. 75 10/10/1997 10/10/1997	Dr. Joseph G. King		Diffuse patchy pulmonary parenchymal consolidation is noted. This may represent ARDS and/or diffuse pneumonia. A pneumothorax is noted at the lung apex of the left and appears small.

Date of X-ray/ reading	Readers' Qualifications	Reading and Film Quality	Result Concerning Presence of Pneumoconiosis
DX 15, p. 74 10/10/1997 10/10/1997	Dr. Joseph G. King		A small left-sided pneumothorax is present. There is noted an increase in extensive patchy bilateral pulmonary parenchymal consolidation consistent with ARDS and/or diffuse pneumonia.
DX 15, p. 77 10/11/1997 10/11/1997	Dr. Joseph G. King		Slight clearing of the extensive patchy bilateral pulmonary parenchymal consolidation. Differential considerations include ARDS and/or diffuse pneumonia. A definite pneumothorax on the left is no longer visualized.
DX 15, p. 76 10/11/1997 10/11/1997	Dr. Joseph G. King		No pneumothorax is seen. Extensive diffuse bilateral pulmonary parenchymal consolidation is again identified, however, appears slightly improved.
DX 15, p. 83 10/15/1997 10/15/1997	Dr. John H. Woodring		Small left apical pneumothorax.
DX 15, p. 88 10/17/1997 10/17/1997	Dr. James L. Buck		Partial improvement in bilateral predominantly interstitial infiltrates and adenopathy. No evidence of abscess.

Date of X-ray/ reading	Readers' Qualifications	Reading and Film Quality	Result Concerning Presence of Pneumoconiosis
DX 15, p. 84 10/17/1997 10/17/1997	Dr. Richard Coleman		No significant change in the diffuse air space disease, right greater than left. No change in the small apical pneumothorax on left.
DX 15, p. 89 10/18/1997 10/18/1997	Dr. William J. Vanarthos		No interval changes observed in the left apical pneumothorax. No interval change in the pulmonary interstitial edema.
DX 15, p. 92 10/20/1997 10/20/1997	Dr. James C. Reed		The miner is status post left-sided thoracotomy. No change in the previously identified right-sided pulmonary edema. Emphysematous changes of the left lung—unchanged from prior examination.
DX 15, p. 90 10/21/1997 10/21/1997	Dr. James C. Reed		Persistent left lower lobe atelectasis. Interval development of small left apical pneumothorax.
DX 15, p. 91 10/22/1997 10/22/1997	Dr. James L. Buck		No significant change in the bilateral interstitial infiltrates of the small left apical pneumothorax.
DX 15, p. 93 10/23/1997 10/23/1997	Dr. John H. Woodring		No significant change in the asymmetric idiopathic pulmonary fibrosis, worse on the right.
DX 15, p. 95 10/24/1997 10/24/1997	Dr. James C. Reed		No significant change in the asymmetric pulmonary fibrosis, right greater than left.

Date of X-ray/ reading	Readers' Qualifications	Reading and Film Quality	Result Concerning Presence of Pneumoconiosis
DX 15, p. 94 10/24/1997 10/24/1997	Dr. James C. Reed		No pneumothorax identified. No significant change in the diffuse interstitial lung disease with increased lucency of the left upper lobe, likely an area of emphysematous change.
DX 15, p. 97 10/26/1997 10/26/1997	Dr. James C. Reed		Internal development of a right-sided pneumothorax. No significant change in the diffuse, severe, fibrotic changes of the lungs with relative area of increased lucency of the left apex, likely representing an area of emphysematous change.

Pulmonary Function Test

Pulmonary function tests (PFT) are performed to measure obstruction in the airways of the lungs and the degree of impairment of pulmonary function. If there is greater resistance to the flow of air, there is more severe lung impairment. The studies range from simple tests of ventilation to very sophisticated examinations requiring complicated equipment. The most frequently performed tests measure forced vital capacity (FVC), forced expiratory volume in one-second (FEV_1) and maximum voluntary ventilation (MVV). The quality standards for PFTs are found at 20 C.F.R. § 718.103 (2000) and Appendix B. The following chart summarizes the results of the PFTs available in this case. "Pre" and "post" refer to administration of bronchodilators. If only one figure appears, bronchodilators were not administered. In a "qualifying" pulmonary test, the FEV_1 must be equal to or less than the applicable values set forth in the tables in Appendix B of Part 718, and either the FVC or MVV must be equal to or less than the applicable table value, or the FEV_1/FVC ratio must be 55% or less. 20 C.F.R. § 718.204(b)(2)(i) (2000).

Ex. No. Test Date Physician	Age Height	FEV_1 Pre-/ Post	FVC Pre-/ Post	FEV_1/ FVC Pre-/ Post	MVV Pre-/ Post	Qualify?	Physician Impression
DX 14, p. 6 06/12/1992 Vuskovich (with medical report)	53/ 182cm[7]	2.56	3.03	84%	Deferred	No	Able to follow directions. Poor cooperation. Invalid studies due to poor effort.
DX 48, p. 63 09/09/1997	59/ 73"	1.40	1.56	90%	Not reported.	Yes	No comments. Only one tracing.
DX 48, p. 31 09/24/1997	58/ 73"	1.44	1.70	85%	Not reported.	Yes	No comments. Only one tracing.

Arterial Blood Gas Studies

Arterial blood gas (ABG) studies are performed to measure the ability of the lungs to oxygenate blood. A defect will manifest itself primarily as a fall in arterial oxygen tension either at rest or during exercise. The blood sample is analyzed for the percentage of oxygen (PO_2) and the percentage of carbon dioxide (PCO_2) in the blood. A lower level of oxygen (O_2) compared to carbon dioxide (CO_2) in the blood indicates a deficiency in the transfer of gases through the alveoli which may leave the miner disabled. The quality standards for arterial blood gas studies are found at 20 CFR § 718.105 (2004). The following chart summarizes the arterial blood gas studies available in this case. A "qualifying" arterial gas study yields values which are equal to or less than the applicable values set forth in the tables in Appendix C of Part 718. If the results of a blood gas test at rest do not satisfy Appendix C, then an exercise blood gas test can be offered. Tests with only one figure represent studies at rest only. Exercise studies are not required if medically not advisable. 20 CFR § 718.105(b) (2004).

[7] 182 cm x 0.3937008 = 71.7 inches. I must first resolve the height discrepancy recorded on the pulmonary function tests. *Protopappas v. Director, OWCP*, 6 B.L.R. 1-221 (1983). Claimant's average reported height is 72.56 inches, which is not a value listed in the regulatory tables. The closest values in inches listed in the regulatory tables are 72.4 and 72.8. I am unable to round up to 72.8 inches and, therefore, find that Claimant's height is 72.4 inches for the purpose of determining qualification under the regulatory tables.

Exhibit Number	Date	Physician	PCO_2 at rest/ exercise	PO_2 at rest/ exercise	Qualify?	Physician Impression
DX 13, p. 60	05/03/1994	Dr. S.J. King	35.9	85.7	No	
DX 13, p. 6	09/06/19997	Dr. William Munro	34.1	66.4	No	
DX 48, pp. 27, 61	09/07/1997	Dr. T. Shawn Caudill	25	61	Yes	

Biopsy and Autopsy Evidence

Dr. Eun Y. Lee

Dr. Lee, from the University of Kentucky Medical Center, performed Miner's lung biopsy on October 9, 1997, and submitted his report on October 14, 1997. DX 36. Dr. Lee's diagnosis was "lung, left lower lobe and left upper lobe, wedge biopsies: consistent with usual interstitial pneumonia (UIP)." The "working" diagnosis was pulmonary fibrosis. Dr. Lee analyzed two specimens, one of the left lower lobe of the lung and one of the left upper lobe of the lung. He did not identify any lesions on gross examination. On microscopic examination, Dr. Lee found lung parenchyma with interstitial inflammatory and fibrosing process. He found marked regional variations in the degree of infiltrate and fibrosis. Dr. Lee noted marked acute and chronic inflammation and destruction of the bronchioles. He stated that in some areas the degree of fibrosis is quite extensive and that the lung is honeycombed. He did not see any granulomas. He found no evidence of malignancy, vasulitis, or eosinophilic granuloma. Dr. Lee concluded that the overall findings were consistent with usual interstitial pneumonia (UIP)/idiopathic pulmonary fibrosis. Two other doctors, Drs. Yoneda and Lower, saw this case and concurred with Dr. Lee's interpretation.

Dr. Richard L. Naeye

Dr. Naeye, Professor of Pathology at The Pennsylvania State University College of Medicine, is Board-certified in anatomic and clinical pathology. DX 34, DX 33. Dr. Naeye received for review a surgical pathology report and four slides with Miner's lung tissue removed by surgery. DX 34. He submitted a written report dated June 14, 1998. He noted that the clinical diagnosis was interstitial pneumonia and that Miner died with a clinical diagnosis of respiratory failure due to idiopathic pulmonary fibrosis. Chest x-rays showed interstitial disease in both lungs. Dr. Naeye also received Miner's medical records from Pikeville Methodist Hospital, University of Kentucky Medical Center, and multiple physicians. Id. He noted that chronic interstitial pulmonary fibrosis first appeared on Miner's chest x-ray in 1978, again in 1983, and later spread to all of Miner's lungs.

On examination of Miner's lung tissue, Dr. Naeye found a very small amount of black pigment free in the tissues adjacent to small airways and just beneath the pleura. He noted that there were large amounts of rather dense fibrous tissue completely replacing lung tissue in many areas and smaller foci of such fibrosis at other sites. The first described area has a honeycomb appearance with bronchioles embedded in the dense fibrous tissue. Smaller foci of interstitial fibrosis and collections of lymphocytes and other chronic inflammatory cells are present in the parenchyma. One small airway is filled with mucous and acute inflammatory cells.

Dr. Naeye concluded that Miner's lung tissue available for analysis did not have any findings of pneumoconiosis. The very small amounts of black pigment did not have any accompanying focal emphysema, tiny birefringent crystals, or related fibrous tissue, which are the minimal findings necessary to make a diagnosis of simple pneumoconiosis. Dr. Naeye stated that where the fibrous tissue and black pigment are together in the same location in Miner's lungs that the fibrous tissue extends far beyond the black pigment so the relationship between the two is fortuitous rather than cause and effect. Dr. Naeye also noted that none of the chest x-ray reports or reports of clinical findings had evidence that the Miner suffered from pneumoconiosis. Dr. Naeye opined that Miner died of a non-occupational disorder, interstitial pulmonary fibrosis that began in 1978 and advance over the years to a severe condition at the time of Miner's death. Dr. Naeye concluded that Miner's disease was completely unrelated to anything produced by exposure to coal mine dust and that Miner would have died at the same time and in the same way even if he had never mined coal. Dr. Naeye stated that it was "noteworthy that this disease progressed year by year and simple coal workers' pneumoconiosis does not advance after a bituminous coal worker quits mining coal." He did not know the cause of Miner's lung disease but found that nothing in the case supported a diagnosis of pneumoconiosis.

Dr. Jerome Kleinerman

On June 22, 1998, Dr. Kleinerman, Board-certified in pathological anatomy and clinical pathology, reviewed Miner's medical documents (detailed below) and analyzed two specimens from Miner's lung biopsy on October 9, 1997. DX 37, p. 9-10. Dr. Kleinerman reported the following:

> Slides A1 and A2 hold sections of subpleural lung tissue with virtually no black granular pigment in the interstitial connective tissue. There are small fields of normal alveolarized lung tissue with bronchioles and blood vessels, however the major part of the lung tissue is replaced by thick fibrocollagenous tissue stroma in which are simplified air spaces lined by cuboidal or flattened epithelium. The interstitial stroma is thick and fibrotic and is infiltrated by clusters of plasmacytes, lymphocytes, and a rare neutrophil. Nodular collections of lymphocytes are also present. Within the air spaces are small and focally distributed granulation

> tissue. The bronchioles appear reasonably normal, but the small arteries and arterioles have thick mural walls and narrowed lumens. There is no evidence of simple CWP, no evidence of simple nodular silicosis, and no evidence of complicated pneumoconiosis.
>
> In the sections labeled B1 and B2 a small amount of black granular pigment is present in the subpleural interstitial tissue. The pathological changes observed in the lung tissue of specimen B are similar to those described in specimen A1 and A2. In the sections marked B1 and B2 there are small collections of alveolar macrophates in airspaces, which contain coarse brown-black pigment. There is however no evidence of simple CWP, simple nodular silicosis, or complicated pneumoconiosis in these sections. (Emphasis in original).

Dr. Kleinerman concluded that Miner's pathological changes were classical for the disease Idiopathic Interstitial Pneumonitis and fibrosis. He opined that Miner's lung sections showed no evidence of simple CWP, simple nodular silicosis, or complicated pneumoconiosis. He also determined that CWP was not a substantial contributing cause of his death, because there was no evidence of simple CWP in the lung biopsies.

Dr. P. Raphael Caffrey

Dr. Caffrey, a Board-certified anatomical and clinical pathologist, reviewed Miner's medical records (detailed below) and analyzed Miner's four surgical pathology slides. DX 38. He submitted a written report on June 27, 1998. Dr. Caffrey noted that all four slides showed similar findings. The findings show varying degrees of interstitial inflammatory infiltrate consisting mainly of lymphocytes, some plasma cells and eosinophils, as well as monocytes. He found varying degrees of interstitial fibrosis. Dr. Caffrey saw only a minimal amount of anthracotic pigment subpleurally. He did not identify any granulomas or asbestos. There was no evidence of vasculitis or neoplasia. Dr. Caffrey's final diagnosis was idiopathic interstitial pulmonary fibrosis with usual interstitial pneumonitis (UIP). He noted that usual interstitial pneumonitis can be seen in association with silicosis and/or asbestosis but that Miner did not have histological evidence of silicosis or asbestosis. The biopsies only showed a minimal amount of anthracotic pigment and definitely did not show the necessary findings to make a diagnosis of simple coal workers' pneumoconiosis.

Dr. Grover M. Hutchins

On July 25, 1998, Dr. Hutchins, Board-certified in anatomic pathology, submitted a medical report after he reviewed Miner's medical documents and four histological slides. DX 43. Dr. Hutchins noted Miner's employment history, smoking history, and medical history. He found that all four slides contained lung tissue with a slight amount

of subpleural, perivascular, peribronchial, and interstitial coal dust pigment with associated birefringent silicate-type particles. He did not find any macules, micronodules, macronodules, or lesions of progressive massive fibrosis. He further found no evidence of asbestos bodies or asbestos associated lesions and no silicotic lesions. Dr. Hutchins concluded that coal workers' pneumoconiosis was not present and, therefore, it could not have contributed to or caused Miner's death. He opined that Miner died from a rapidly progressive usual interstitial pneumonia and idiopathic pulmonary fibrosis. He stated that there is no known relationship between usual interstitial pneumonia or idiopathic pulmonary fibrosis and exposure to coal dust or having simple coal workers' pneumoconiosis. Dr. Hutchins opined that Miner would have died at the same time and in the same manner had he never been employed as a coal miner.

Medical Opinions

Medical opinions are relevant to the issues of whether the miner has pneumoconiosis and whether pneumoconiosis was a substantially contributing cause of the miner's death under § 718.205. A determination of the existence of pneumoconiosis may be made if a physician, exercising sound medical judgment, notwithstanding a negative x-ray, finds that the miner suffered from pneumoconiosis as defined in 20 CFR § 718.201. See 20 CFR § 718.202(a)(4). Thus, even if the x-ray evidence is negative, medical opinions may establish the existence of pneumoconiosis. *Taylor v. Director, OWCP*, 9 B.L.R. 1-22 (1986). The medical opinions must be reasoned and supported by objective medical evidence such as blood gas studies, electrocardiograms, pulmonary function studies, physical performance tests, physical examination, and medical and work histories. 20 CFR § 718.202(a)(4). Quality standards for reports of physical examinations are found at 20 CFR § 718.104. The record contains the following medical opinions relating to this case.

Dr. Matt Vuskovich

On June 12, 1992, Dr. Vuskovich examined the miner at the request of attorney Brett D. Davis. DX 14, p. 2. Dr. Vuskovich practices in the fields of occupational medicine and primary care. He is also a B-reader. He took occupational, social, smoking, family, and medical histories. Dr. Vuskovich also performed a physical examination, chest x-ray, pulmonary function study, arterial blood gas study, and an electrocardiogram. He diagnosed the miner with chronic bronchitis, secondary to cigarette smoking. He opined that the miner did not have an occupational lung disease caused by coal mine employment. Dr. Vuskovich also determined that the miner was physically able, from a pulmonary standpoint, to do his usual coal mine employment or comparable and gainful work in a dust-free environment.

Dr. Samuel J. King

Dr. King addressed a letter to "To Whom It May Concern" on November 21, 1996. DX 48, p. 72. He stated that Miner was a patient since 1987. Dr. King treated

Miner for arthritis, ulcer disease, hiatal hernia, and COPD. He noted that chest x-rays have shown chronic diffusive anistial disease. The working diagnoses were (1) chronic pain syndrome, (2) depression, reactive with anxiety, (3) COPD, and (4) history of peptic ulcer disease. He stated that Miner was not released to return to work. The bulk of Dr. King's letter addressed Miner's back injuries.

Dr. King issued a supplemental report on January 2, 1997, addressing Miner's chronic mechanical back pain with lumbosacral strain, degenerative disc disease, and muscle spasms.

Dr. T. Shawn Caudill

Dr. Rush Fisher, an Orthopaedic Surgeon at the University of Kentucky, referred Miner to Dr. Caudill, an Associate Professor of Medicine at University of Kentucky, for a pre-operative evaluation for an upcoming lumbar disc surgery. DX 48, pp. 59-63. Dr. Caudill issued a consultative report dated September 9, 1997. He noted that Miner had relatively recent onset of pulmonary problems. Miner reported that he developed increasing shortness of breath and sputum production three months ago. Miner stated that he was diagnosed with "chronic bronchitis and 'coal miner's pneumoconiosis' with an acute flare-up." Miner's condition originally improved with medication but his symptoms deteriorated on September 6, 1997, and he was treated in the Pikeville emergency room and diagnosed with acute bronchitis with questionable pneumonia. Miner stated that he continued to have severe dyspnea on exertion, copious sputum production, and shortness of breath at rest.

Dr. Caudill reviewed Miner's history. He noted that Miner stopped smoking 5 years prior but had a 27-pack-year history of smoking (1 pack per day). He also reported that Miner was on disability for lung disease. Dr. Caudill reported that miner worked 30-years in the coal mines with long-term exposure to coal dust.

On physical examination, Dr. Caudill found that Miner had mild difficulty breathing with conversation but no distress. Miner frequently coughed and expelled sputum during the examination. Dr. Caudill found diffuse dry rales in all of Miner's lung fields with diminished excursion and scattered rhonchi. Miner's heart sounds were somewhat masked by lung crackles. Dr. Caudill noted that Miner had marked clubbing in his digits in the upper extremities with minimal cyanosis but no edema. Dr. Caudill also performed an EKG, chest x-ray, pulmonary function test, and arterial blood gas study.

Dr. Caudill determined that Miner's pulmonary status was severely compromised. Miner had an ongoing fibrotic process with copious mucus secretions. He gained some relief from his treatment for acute bronchitis. Dr. Caudill worried about Miner's dyspnea at rest, large amounts of sputum production, and clubbing of his digits. Dr. Caudill recommended against elective surgery.

Dr. R. Scott Morehead

Dr. Morehead conducted a Level V New Patient Evaluation on September 23, 1997. DX 48, pp. 14-15. He noted that Miner was referred to him due to respiratory debilitation and probable pulmonary fibrosis. Miner reported a long history of lung disease, 30 years of coal mine employment, and a 27-pack year smoking history. Miner stated that his pulmonary problems worsened over the past 9 months. He complained of a significant cough with at least a half cup full of purulent sputum, worsening dyspnea, and chest pain with exertion. On physical examination, Dr. Morehead found markedly reduced breath sounds with extensive fine inspiratory crackles throughout and including the anterior fields. No wheezing or rhonchi were appreciated. Dr. Morehead stated that the spirometry showed fine to severe restriction with normal FEV_1/FVC ratio. Dr. Morehead concluded that Miner had severe interstitial lung disease that mimics IPF. "Although the findings could be consistent with a number of other things including hypersensitivity pneumonitis, silicosis, asbestosis, the rales and lack of exposure history make me think the IPF is most likely."

Dr. Joshua A. Perper

Dr. Perper, Board-certified in anatomical and surgical pathology and forensic pathology, reviewed the miner's smoking history, drinking history, 20-year clinical history, and death certificate. DX 14a, pp. 1-20 and DX 15, pp. 99-117. On March 10, 1998, Dr. Perper submitted his findings to Ms. Phyllis Sturgill, a Claims Examiner for the U.S. Department of Labor. He noted that the miner's body was not autopsied; therefore, a determination regarding the existence of pneumoconiosis was based only on the clinical data available from the miner's related medical documentation. Dr. Perper stated that a clinical diagnosis of coal workers' pneumoconiosis and related respiratory impairment and significance is generally made on the basis of occupational history, clinical symptomatology, findings on physical examination, spirometry findings, arterial blood gas findings, radiological findings of pulmonary opacities corresponding with pneumoconiotic lesions, biopsy or autopsy findings, and consideration of confounding co-existing health habits or conditions. Dr. Perper concluded that the miner suffered from coal workers' pneumoconiosis based upon the radiological findings of interstitial fibrosis and diffuse nodularity with clinical symptoms and manifestations of restrictive lung disease and COPD. Dr. Perper determined that coal workers' pneumoconiosis was a substantial contributory cause of the miner's death based upon the clinical, radiological, and pulmonary function evidence.

Dr. Bruce C. Broudy

Dr. Broudy is Board-certified in internal medicine and pulmonary medicine; he is also a B-Reader. DX 31, p. 7. Dr. Broudy reviewed Miner's medical records and submitted a written evaluation on June 2, 1998. DX 31, p. 2. Miner's cause of death was listed as respiratory failure due to idiopathic pulmonary fibrosis. *Id*. He noted that the results of Miner's lung biopsy showed idiopathic pulmonary fibrosis with usual interstitial pneumonitis. *Id*. Dr. Broudy stated that this was quite distinct from the finings one would

expect to see in coal workers' pneumoconiosis. *Id*. "Had one arrived at a diagnosis of coal workers' pneumoconiosis, there would have been no need for treatment with corticosteroids or immunosuppressive agents." *Id*. Dr. Broudy disagreed with Dr. Perper's conclusions that Miner had disabling coal workers' pneumoconiosis and that this contributed to his death. DX 31, p. 3. Although Miner had sufficient exposure to coal dust, Dr. Broudy opined that neither Miner's radiographic changes nor the pathological findings were characteristic of coal workers' pneumoconiosis. *Id*. Dr. Broudy concluded that one cannot reasonably make a diagnosis of coal workers' pneumoconiosis because the biopsy evidence did not show progressive massive fibrosis that is seen with complicated pneumoconiosis nor did it show the characteristic lesions of simple pneumoconiosis. DX 31, p. 4. He determined that pneumoconiosis was not a substantial contributing cause of Miner's death because it was not even possible to make a definitive diagnosis of pneumoconiosis. *Id*. However, even if simple pneumoconiosis existed on the biopsy, Dr. Broudy concluded that it would not have contributed to Miner's death. *Id*. Miner's death was due to the progressive usual interstitial pneumonia due to idiopathic pulmonary fibrosis. *Id*. Dr. Broudy opined that it would be highly unusual to have a negative chest x-ray in 1992 per Dr. Vuskovich and then to die of coal workers' pneumoconiosis some 5 years later. *Id*.

Dr. Raghu R. Sundaram

Dr. Sundaram reviewed Miner's medical records and submitted a written evaluation on June 3, 1998. DX 32, p. 2. Dr. Sundaram stated that Miner had idiopathic pulmonary fibrosis. *Id*. He needed to look at the autopsy report of Miner's lungs[8] to rule out pneumoconiosis. *Id*. Dr. Sundaram concluded that idiopathic pulmonary fibrosis and the related complications contributed to Miner's death. *Id*. He further found that Miner's substantial impairment and disease related to idiopathic pulmonary fibrosis and complications led to Miner's demise. *Id*.

Dr. Jerome Kleinerman

Dr. Kleinerman reviewed Miner's medical records since 1977 and issued a written report on June 22, 1998. DX 37. Dr. Kleinerman noted Miner's coal mine employment history of 28-29 years, his cigarette smoking history of 6 cigarettes to 2 packs per day for 20-28 years, his social history, and his medical history. Dr. Kleinerman concluded that Miner had Diffuse Idiopathic Interstitial Fibrosis (DIF) in his lung tissues. He noted that there are many recognized causes of DIF which may indicate rheumatoid lung disease, scleroderma, sarcoidosis, allergic alveolitis, and certain occupational lung diseases such as asbestosis. Dr. Kleinerman determined that there was no evidence of any of these disease states. (Emphasis in original). Dr. Kleinerman stated that the largest group of diffuse interstitial pneumonitis and fibrosis, as many as 50% of the cases, no identifiable causative agent is recognized or defined for the interstitial fibrosis and pneumonitis. He concluded that Miner's diffuse interstitial pneumonitis and fibrosis were idiopathic.

[8] The family declined an autopsy.

Dr. Kleinerman refuted each of Dr. Perper's reasons for determining that Miner suffered from pneumoconiosis. He even noted that Dr. Perper incorrectly summarized that Miner did not have a lung biopsy. Dr. Kleinerman stated that Dr. Perper's "pseudo criteria" were neither objective or nor specific and did not justify a finding of CWP. Finally, Dr. Kleinerman found that Dr. Perper's references also did not provide evidence that Miner's lung disease was CWP.

Dr. Kleinerman concluded that Miner's lung biopsy demonstrated with definite and conclusive objectivity that he did not have simple or complicated CWP in the lung tissue. However, there were clear and distinctive pathological lesions of idiopathic diffuse interstitial pneumonitis and fibrosis. Dr. Kleinerman opined that these were not in any way related to or caused by coal mine dust inhalation.

Dr. P. Raphael Caffrey

Dr. Caffrey reviewed Miner's medical records and chest x-rays from the University of Kentucky Medical Center and the Pikeville Methodist Hospital. DX 38. He also reviewed reports from Drs. Broudy and Sundaram. On June 27, 1998, Dr. Caffrey stated that he "definitely cannot make a diagnosis of coal workers' pneumoconiosis or any other occupational pneumoconiosis." He noted that the chest x-ray interpretations do not make a diagnosis of pneumoconiosis. Dr. Caffrey stated, "I see nothing in these medical records or the surgical pathology slides where there is even a questionable diagnosis of coal workers' pneumoconiosis." Because there was no autopsy, Dr. Caffrey concluded that it was unknown if pneumoconiosis was in Miner's right lung. However, if any disease of coal workers' pneumoconiosis would have been in the right lung it would have been mild because it did not show up on chest x-ray. Dr. Caffrey determined that even if there were pneumoconiosis in the right lung, it would have been, at most, simple pneumoconiosis and would not have caused or contributed to Miner's disease of idiopathic pulmonary fibrosis or caused or contributed to his death.

Dr. Caffrey described idiopathic interstitial fibrosis with usual interstitial pneumonitis. He stated that this is a spectrum of disease, the exact etiology, which in most cases is unknown. Some reports indicated that this disease occurs in patients with liver disorders, those with genetic factors, or those who smoke. Dr. Caffrey stated that this is a disease with an insidious onset that can take a fairly progressive course over a few years and that may or may not respond to steroid therapy. Dr. Caffrey noted that Miner did not respond to the steroid therapy.

Dr. Caffrey opined that there was no evidence in the Miner's medical records or surgical pathology biopsy slides that Miner had coal workers' pneumoconiosis or any other occupational pneumoconiosis. He concluded that Miner suffered from idiopathic interstitial pulmonary fibrosis with usual interstitial pneumonitis which is a progressive, fatal pulmonary disease that has no relationship to his employment in the coal mining industry.

On July 15, 1998, Dr. Caffrey issued a supplemental report after he reviewed additional medical documents and reports by other physicians. DX 42. Dr. Caffrey continued to opine that Miner did not have coal workers' pneumoconiosis or any other occupational pneumoconiosis. He concluded that Miner had idiopathic interstitial fibrosis which was not caused by his employment in the coal mining industry.

Dr. Caffrey went on to disagree with Dr. Perper's conclusions in his report dated March 10, 1998. He stated, "I have no idea how Dr. Perper arrived at the diagnosis of coal workers' pneumoconiosis and in his 20-page report, it is my opinion that he definitely does not say how he arrived at it."

Dr. Thomas M. Jarboe

Dr. Jarboe, a B-Reader who is Board-certified in internal medicine, reviewed Miner's medical records and chest x-rays and Dr. Perper's report and submitted his own written evaluation dated July 10, 1998. DX 40. Dr. Jarboe concluded that Miner did not have coal workers' pneumoconiosis. The most compelling evidence against the diagnosis was the biopsy results that showed a usual interstitial pneumonia with associated pulmonary fibrosis. Dr. Jarboe did not see any mention whatsoever any of the characteristics of coal workers' pneumoconiosis such as coal macules, silica, coal particles, or focal emphysema.

Dr. Jarboe opined that Miner exhibited all the classic hallmarks of usual interstitial pneumonia: progressive dyspnea, progressive advancement of the interstitial process on the chest films, fine crackling rales on examination, and marked clubbing of the digits. Dr. Jarboe stated that it would be very unlike coal workers' pneumoconiosis to produce any of these changes. He specifically noted that Dr. Perper's rationale that pneumoconiosis developed after Miner left the mines had nothing to do with this claim, because Miner did not have progressive massive fibrosis or any suggestion of the same.

Dr. Jarboe concluded that coal workers' pneumoconiosis was not a substantially contributing cause of Miner's death, because there was no evidence of pneumoconiosis. Miner suffered from a pulmonary condition called usual interstitial pneumonia which is an inflammatory reaction in the alveolar spaces which eventually results in fibrosis and scarring of the lungs. Dr. Jarboe did not believe that coal dust inhalation caused usual interstitial pneumonitis as it was classically presented in Miner's case. He opined that Miner died from respiratory failure caused by usual interstitial pneumonitis with associated fibrosis and that Miner would have died of the same cause and at the same time whether or not he had ever worked as a coal miner.

Dr. Jarboe opined that Miner's most prominent lung condition was usual interstitial pneumonia/pulmonary fibrosis. The cause was not clear and is, often, not known. He stated that it could be associated with viral infections and other causes. Dr. Jarboe also opined that Miner had chronic obstructive lung disease with an asthmatic component caused by his 27-year smoking history. Miner also had recurrent

bouts of purulent bronchitis/pneumonitis most likely caused by the underlying lung damage from the UIP and COPD.

Dr. Peter Tuteur

Dr. Tuteur, Board-certified in internal medicine and pulmonary disease, reviewed Miner's medical records and issued a consultative report dated August 15, 2005. EX 1. He noted Miner's extensive coal mine employment history and smoking history. He detailed Miner's history of progressive pulmonary problems. Dr. Tuteur reported that Miner died with and because of usual interstitial pneumonitis, its sequelae, and treatment. Dr. Tuteur did not find any convincing supportive information for the diagnosis of coal workers' pneumoconiosis or any other coal mine dust related disease process. Based on the totality of all available medical data, Dr. Tuteur opined that Miner did not have coal workers' pneumoconiosis of sufficient severity and profusion to produce clinical symptoms, physical examination abnormalities, impairment of pulmonary function, radiographic change at either the level of the standard chest x-ray or the CT, or to produce pathologic change identifiable at the time of histologic examination of the lung tissue obtained during lung biopsy. However, Dr. Tuteur did agree that Miner had a primary pulmonary disease process. The process was progressive usually interstitial pneumonitis (idiopathic pulmonary fibrosis with inflammation), a condition unrelated to, not aggravated by, and not caused by either the inhalation of coal mine dust or the development of coal workers' pneumoconiosis. Dr. Tuteur opined that it was this condition that eventually rendered Miner disabled and caused his death. Dr. Tuteur stated that Miner's death was in no way caused by, influenced by, or hastened by either the inhalation of coal mine dust or the development of coal workers' pneumoconiosis. Dr. Tuteur determined that Miner's clinical course, his health problems, and his ultimate demise would have been no different even if he had never worked in the coal mine industry.

He noted that his conclusions were supported by Miner's extensive data and were in agreement with virtually all the medical reviewers except Dr. Perper. Dr. Tuteur found that there was no meaningful supportive information for Dr. Perper's isolated and unique conclusion with respect to the presence of coal workers' pneumoconiosis in Miner's lungs.

Dr. Joshua A. Perper

Dr. Perper, Board-certified in anatomical and surgical pathology and forensic pathology, issued a consultative report dated June 5, 2005. CX 1. Dr. Perper reviewed 20 years' worth of Miner's medical history; Miner's lung biopsy slides; opinion reports by Drs. Broudy, Caffrey, Caudill, Hutchins, Kleinerman, Jarboe, Morehead, and Naeye; and Decisions and Orders by the Administrative Law Judges and the Benefits Review Board.

Upon reviewing the biopsy slides, Dr. Perper diagnosed "(1) coal workers' pneumoconiosis, interstitial pulmonary fibrosis type, (2) centrilobular emphysema,

marked and honeycombing, (3) sclerosis of intrapulmonary blood vessels, severe, consistent with pulmonary hypertension and core pulmonale." Dr. Perper determined that Miner had pulmonary interstitial fibrosis with the presence of slight to moderate deposition of anthracotic pigment and presence of birefringent silica crystals, consistent with the atypical pattern of interstitial type coal workers' pneumoconiosis. He concluded that Miner developed pneumoconiosis as a result of his occupational exposure to coal dust of at least 22 years and that it was unreasonable to argue that Miner's pulmonary interstitial fibrosis was idiopathic, *i.e.* of unknown original. Dr. Perper indicated that scientific literature has recognized exposure to coal dust as a causal factor of centrilobular emphysema along; he also acknowledged that centrilobular emphysema can be caused by cigarette smoking. Dr. Perper concluded that pneumoconiosis resulted in Miner's total and irreversible respiratory disability and was the primary cause of his death. Dr. Perper found flaws in the reports of Drs. Broudy, Caffrey, Kleinerman, Jarobe, and Naeye.

Medical Treatment Records

Miner was treated and admitted a number of times to the Methodist Hospital of Kentucky and the Pikeville Methodist Hospital from 1977 to 1997. DX 13. His dates of treatments and diagnoses are as follows: February 13, 1977, to February 17, 1977, for acute tracheo-bronchitis; February 18, 1977, to February 26, 1977, for pneumonia; September 30, 1977, to October 5, 1977, for pneumonia with basilar atelectasis of both lobes; March 4, 1979, for back pain; May 15, 1979, for pain in his left elbow; May 17, 1979, for pain in his left elbow; October 24, 1979, for x-ray of his right forearm following an accident; June 7, 1980, for a head injury; October 6, 1982, for chest pain; September 17, 1983, for the flu; January 1, 1984, for chest pain; May 2, 1984, to May 12, 1984, for back pain; June 16, 1985, to June 30, 1985, for back and hip problems; April 20, 1986, for pain in his right knee following a fall. April 25, 1988, to April 29, 1988, for abdominal pain, acute duodenal ulcer, hiatal hernia with reflux, bile reflux, diverticular disease, and left subscapular bursitis; November 20, 1988, for a three-car accident; July 18, 1989, for injury to head from a rock fall; February 11, 1990, for acute back pain following an injury at work; December 4, 1990, for low back pain; July 16, 1991, surgery for peptic ulcer disease; March 28, 1991, surgery for gastritis, acute duodenal ulcer, and duodenitis; March 17, 1994, for back pain; October 28, 1994 for hiatal hernia and chronic gastritis; November 24, 1995, for disc disease following a trip and fall at work; July 22, 1996, for chest pain; December 2, 1996, for a fall from a high wall into the road; January 31, 1997, for acute bronchitis; February 14, 1997, for a URI; March 17, 1997, for lower back pain; April 9, 1997, for back pain; May 14, 1997, for pain in lower back, left hip, and left leg; May 22, 1997, for pain in lower back and left leg; May 28, 1997, for pain in lower back and left leg; June 11, 1997, for follow-up of lower back and left leg pain; and July 10, 1997, for back and leg pain. Only the February 18, 1977, hospital report mentions that Miner had been previously treated for coal workers' pneumoconiosis.

Miner was treated in numerous times in 1987 for back pain and left leg pain. DX 48. He also had surgery on October 8, 1987 for a herniated nucleus pulposus L4-5, degenerative bulging disc L5-S1. DX 48, p. 121.

Miner was treated at the Methodist Hospital of Kentucky from April 25, 1988 to April 29, 1988, for abdominal pain, acute duodenal ulcer, hiatal hernia with reflux, bial reflux, diverticular disease, and left subscapular bursitis. DX 48, p. 117.

On November 9, 1993, Miner was admitted to the Methodist Hospital of Kentucky. DX 48, pp. 94, 91. He had upper respiratory symptoms for three weeks prior to admission. On the day of admission, he was short of breath, had audible wheezes, and coughed to the point of vomiting. During his hospital stay, he required steroids because of his persistent wheezing. He was discharged on November 14, 1993. His discharge diagnoses were (1) acute asthmatic bronchitis, (2) chronic obstructive pulmonary disease, (3) hemoptysis, and (4) mechanical low back pain.

Miner was admitted to the Methodist Hospital of Kentucky from May 2, 1994, to May 6, 1994, for bilateral pneumonia, chronic obstructive pulmonary disease, and degenerative osteoarthritis, and chronic mechanical back pain. DX 13, pp. 56-64; DX 48, pp. 77, 84. Dr. S. J. King reported that Miner had upper respiratory symptoms with increased shortness of breath, productive cough of green phlegm, some blood-tinged sputum, fever, and chills. Dr. King also noted that Miner was a coal miner and smoked one pack of cigarettes per day. On physical examination, Dr. King found bilateral rales with expiratory wheezes of moderate pitch, no rubs, in the chest.

Dr. Leon B. Briggs treated Miner at the Methodist Hospital of Kentucky on April 9, 1997, for his lumbar back pain. DX 48, p. 69. It is significant to note that Miner denied the use of tobacco.

Miner's treatment notes are included in the record at July 22, 1985, to July 24, 1997. DX 48, pp. 36-58. He was treated for back pain, injuries, knee pain, inflamed chest with muscle spasms, colds, tendonitis, abdominal pain, stomach problems, ulcers, COPD, and reactive depression secondary to pain.

On September 6, 1997, Dr. William Munro treated Miner in the Emergency Room at the Methodist Hospital of Kentucky. DX 13, p. 2; DX 48, p. 67. Miner's chief complaint was shortness of breath. Dr. Munro performed a physical examination which revealed shortness of breath, bilateral crepitus on auscultation of the pulmonary fields, a chronic hacking cough, and green sputum production. Dr. Munro diagnosed Miner with bronchitis with exacerbation of chronic obstructive pulmonary disease.

Miner was admitted to the University of Kentucky Medical Center on October 9, 1997, for thoracoscopy with lung biopsy which showed usual interstitial pneumonitis with interstitial pulmonary fibrosis. DX 15, p. 8. Miner was dependent on a ventilator for the course of his hospital stay and required increased amounts of oxygen. DX 15, p.8. Claimant and her two sons requested that Miner's life support be removed. DX 15, p.8. Miner had no spontaneous respirations and no pulse. DX 15, p.8. He passed away on October 26, 1997, at 10:34 p.m. DX 15, p. 8. The family declined an autopsy. DX 15, p. 68.

DISCUSSION AND APPLICABLE LAW

Existence of Pneumoconiosis

The regulations define pneumoconiosis broadly:

> (a) For the purpose of the Act, "pneumoconiosis" means a chronic dust disease of the lung and its sequelae, including respiratory and pulmonary impairments, arising out of coal mine employment. This definition includes both medical, or "clinical", pneumoconiosis and statutory, or "legal", pneumoconiosis.
>
> (1) Clinical Pneumoconiosis. "Clinical pneumoconiosis" consists of those diseases recognized by the medical community as pneumoconioses, *i.e.*, the conditions characterized by permanent deposition of substantial amounts of particulate matter in the lungs and the fibrotic reaction of the lung tissue to that deposition caused by dust exposure in coal mine employment. This definition includes, but is not limited to, coal workers' pneumoconiosis, anthracosilicosis, anthracosis, anthrosilicosis, massive pulmonary fibrosis, silicosis or silico-tuberculosis, arising out of coal mine employment.
>
> (2) Legal Pneumoconiosis. "Legal pneumoconiosis" includes any chronic lung disease or impairment and its sequelae arising out of coal mine employment. This definition includes, but is not limited to any chronic restrictive or obstructive pulmonary disease arising out of coal mine employment.
>
> (b) For purposes of this section, a disease "arising out of coal mine employment" includes any chronic pulmonary disease or respiratory or pulmonary impairment significantly related to, or substantially aggravated by, dust exposure in coal mine employment.
>
> (c) For purposes of this definition, "pneumoconiosis" is recognized as a latent and progressive disease which may first become detectable only after the cessation of coal mine dust exposure.

20 CFR § 718.201.

20 CFR § 718.202(a) provides that a finding of the existence of pneumoconiosis may be based on evidence from a (1) chest x-ray, (2) biopsy or autopsy, (3) application of the presumptions (not applicable here) described in Sections 718.304, 718.305, or 718.306, or (4) a physician exercising sound medical judgment based on objective medical evidence and supported by a reasoned medical opinion. In order to determine whether the evidence establishes the existence of pneumoconiosis, I must consider the chest x-rays, the biopsy evidence, and medical opinions – the three categories of evidence applicable in this case. As this claim is governed by the law of the Sixth Circuit, the Claimant may establish the existence of pneumoconiosis under any one of

the alternate methods set forth at Section 718.202(a). *See Furgerson v. Jericol Mining, Inc.*, 22 B.L.R. 1-216 (2002) (*en banc*).

Pneumoconiosis is a progressive and irreversible disease. *Woodward v. Director, OWCP*, 991 F.2d 314, 320 (6th Cir. 1993). As a general rule, therefore, more weight is given to the most recent evidence. See *Mullins Coal Co. of Virginia v. Director, OWCP*, 484 U.S. 135, 151-152 (1987); *Eastern Associated Coal Corp. v. Director, OWCP*, 220 F.3d 250, 258-259 (4th Cir. 2000); *Crace v. Kentland-Elkhorn Coal Corp.*, 109 F.3d 1163, 1167 (6th Cir. 1997); *Rochester & Pittsburgh Coal Co. v. Krecota*, 868 F.2d 600, 602 (3rd Cir. 1989); *Stanford v. Director, OWCP*, 7 B.L.R. 1-541, 1-543 (1984); *Tokarcik v. Consolidated Coal Co.*, 6 B.L.R. 1-666, 1-668 (1983); *Call v. Director, OWCP*, 2 B.L.R. 1-146, 1-148-1-149 (1979). This rule is not to be mechanically applied to require that later evidence be accepted over earlier evidence. *Woodward*, 991 F.2d at 319-320; *Adkins v. Director, OWCP*, 958 F.2d 49 (4th Cir. 1992); *Burns v. Director, OWCP*, 7 B.L.R. 1-597, 1-600 (1984).

Analysis of X-ray Evidence

The chest x-ray evidence in this claim is extensive. There are 59 interpretations of 35 chest x-rays dated February 14, 1977; February 18, 1977; February 23, 1977; September 30, 1977; October 5, 1977; November 4, 1977; October 6, 1982; September 17, 1983; January 4, 1984; April 22, 1987; July 15, 1991; June 12, 1992; March 17, 1993; November 9, 1993; May 2, 1994; May 5, 1994; May 10, 1994; November 24, 1995; July 18, 1996; January 31, 1997; September 6, 1997; September 24, 1997; September 25, 1997; October 9, 1997; October 10, 1997; October 11, 1997; October 15, 1997; October 17, 1997; October 18, 1997; October 20, 1997; October 21, 1997; October 22, 1997; October 23, 1997; October 24, 1997; and October 26, 1997. It is, however, important to note that the majority of these interpretations do not use the ILO-UICC classification form. I am permitted to consider interpretations that are in substantial compliance with the regulations notwithstanding the lack of classification. Conversely, I may determine that interpretations are not in substantial compliance with the regulations if the physician's interpretation was for reasons unrelated to diagnosing the existence of pneumoconiosis. In some circumstances, it is proper for the administrative law judge to infer that an interpretation, which does not mention the presence of pneumoconiosis, as negative. *Marra v. Consolidation Coal Co.*, 7 B.L.R. 1-216 (1984). When an x-ray is not classified and makes no mention of pneumoconiosis, the administrative law judge has discretion to infer whether or not the x-ray is negative for pneumoconiosis. *Billings v. Harlan #4 Coal Co.*, BRB No. 94-3721 BLA (June 19, 1997) (*en banc*) (unpublished).

In this case, the three physicians who reviewed the February 14, 1997 film classified it as unreadable. Therefore, I accord this film no weight.

Four physicians interpreted the September 17, 1983, film. Dr. Poulos' interpretation was apparently for purposes other than the diagnosis of pneumoconiosis, was not in substantial compliance with the regulations, and, therefore, is accorded little

probative weight. Meanwhile, both Drs. Broudy and Jarboe, a B-reader, found this film negative for pneumoconiosis, Category 0. Dr. Sargent, a Board-certified radiologist and B-reader, determined that the film was unreadable because it was a copy. I find that the film dated September 17, 1983, is negative for pneumoconiosis.

The July 15, 1991 film was also interpreted by four physicians. Dr. Halbert's interpretation does not mention the presence or absence of pneumoconiosis and is not in substantial compliance with the regulations. Therefore, I accord his interpretation little probative weight. Dr. Broudy and Dr. Jarboe, a B-reader, both found this film to be negative for pneumoconiosis under the ILO classification system. Dr. Sargent determined that this film was unreadable because it was a copy. I find that this film is negative for pneumoconiosis.

Dr. Vuskovich, a B-reader, was the only physician to interpret the June 12, 1992 chest x-ray. His interpretation complies with the regulations. Although he remarked that there was diffuse interstitial fibrosis in the lower zones, he determined that the film was negative for pneumoconiosis. Thus, I find that the June 12, 1992, film is negative for pneumoconiosis.

Drs. Halbert, Sargent (a Board-certified radiologist and B-reader), Broudy, and Jarboe (B-reader) interpreted the chest x-ray dated November 9, 1993. Dr. Halbert's interpretation does not substantially comply with the quality standards and is afforded little probative weight. Drs. Broudy and Jarboe both found that the film was negative, Category 0, for pneumoconiosis. Dr. Sargent concluded that the film was unreadable because it was a copy. I find that the November 9, 1993, film is negative for pneumoconiosis.

Drs. Poulos and Sundaram interpreted the chest x-ray dated May 10, 1994. Dr. Poulos' interpretation fails to meet the quality standards and, thus, is accorded little probative weight. Dr. Sundaram did not make any markings on his ILO classification form regarding the presence of pneumoconiosis. Rather, he wrote "idiopathic pulmonary fibrosis" in the section for other comments. As such, I find that the May 10, 1994, film is negative for pneumoconiosis.

Four physicians interpreted the January 31, 1997, chest x-ray. Dr. Halbert did not comment on the presence or absence of pneumoconiosis. His interpretation does not substantially comply with the quality standards and is entitled to little probative weight. Dr. Jarboe, a B-reader, found the film negative, Category 0/0, for pneumoconiosis. Dr. Broudy found the film negative for pneumoconiosis but thought that the film quality was quite poor. Dr. Sargent, a Board-certified radiologist and B-reader, found that the film was unreadable because it was a copy. I find that the January 31, 1997, film is negative for pneumoconiosis.

Although Dr. Jarboe, a B-reader, found the September 6, 1997, film to be negative for pneumoconiosis, he also concluded that the film quality was poor. Drs. Sargent and Broudy both found that the film was unreadable because it was a copy.

Dr. Poulos's interpretation was not in substantial compliance with the quality standards. I, therefore, accord the September 6, 1997, film no weight in this analysis.

The films dated February 18, 1977; February 23, 1977; September 30, 1977; October 5, 1977; November 4, 1977; October 6, 1982; January 4, 1984; April 22, 1987; March 17, 1993; May 2, 1994; May 5, 1994; November 24, 1995; September 25, 1997; October 9, 1997; October 10, 1997; October 11, 1997; October 15, 1997; October 17, 1997; October 18, 1997; October 20, 1997; October 21, 1997, October 22, 1997; October 23, 1997; October 24, 1997; and October 26, 1997, were not classified and do not refer to the presence or absence of pneumoconiosis. I accord these films little probative weight because they are not in substantial compliance with the regulations.

I have found that the chest x-ray interpretations that were in substantial compliance with the quality standards set forth in the regulations were negative for pneumoconiosis. I conclude that the Claimant has not established the existence of pneumoconiosis by virtue of the x-ray evidence. Hence, I find that ALJ Phalen did not make a mistake in determination of fact.

Analysis of Biopsy Evidence

A biopsy or autopsy conducted and reported in compliance with §718.106 may constitute the basis for a finding of the existence of pneumoconiosis. 20 C.F.R. §718.202(a)(2). Section 718.106 sets forth the quality standards for autopsies and biopsies. However, the Board, in *Dillon v. Peabody Coal Co.*, 11 B.L.R. 1-113 (1988), held that the quality standards are not mandatory and failure to comply with the standards goes to the reliability and weight of the evidence.

Miner underwent a lung biopsy on October 9, 1997, shortly before his death. Dr. Lee performed the biopsy and five other physicians completed independent reviews of the histological slides. Dr. Lee did not report any evidence of black pigmentation, but he did find evidence of pulmonary fibrosis which was quite extensive in some places. Dr. Lee concluded that Miner had usual interstitial pneumonia/idiopathic pulmonary fibrosis.

Dr. Naeye found a very small amount of black pigment free in the tissues adjacent to the small airways and just beneath the pleura. Dr. Naeye also noted that there were large amounts of rather dense fibrous tissue completely replacing lung tissue in many areas and smaller foci of such fibrosis at other sites. Nevertheless, Dr. Naeye concluded that Miner did not have pneumoconiosis because the very small amounts of black pigment did not have any accompanying focal emphysema, tiny birefringent crystals, or related fibrous tissue, which were the minimal findings necessary to make a diagnosis of simple pneumoconiosis. Dr. Naeye found that the relationship between the black pigment and the fibrosis was fortuitous rather than causative because the fibrous tissue extended far beyond the black pigment found in Miner's lungs.

Dr. Kleinerman found virtually no black granular pigment in the interstitial connective tissue but there was thick fibrocollagenous tissue in two lung sections. In the

other two lung sections, there was a small amount of black granular pigment in the subpleural interstitial tissue with similar pathological changes seen in the first two lung sections. However, Dr. Kleinerman concluded that Miner's pathological changes were classical for the disease idiopathic interstitial pneumonitis and fibrosis. He found no evidence of coal workers' pneumoconiosis, simple nodular silicosis, or complicated pneumoconiosis.

Dr. Caffrey found varying degrees of interstitial fibrosis in Miner's lungs but saw only a minimal amount of anthracotic pigment subpleurally. He diagnosed Miner with idiopathic interstitial pulmonary fibrosis and usual interstitial pneumonitis. He stated that the biopsies only showed a minimal amount of anthracotic pigment and definitely did not show the necessary findings to make a diagnosis of simple coal workers' pneumoconiosis.

Dr. Hutchins found that all four slides from Miner's lung biopsy contained lung tissue with a slight amount of subpleural, pervascular, peribronchial, and interstitial coal dust pigment with associated birefringent silicate type particles. He did not find evidence of progressive massive fibrosis, silicotic lesions, or asbestos bodies. Dr. Hutchins concluded that Miner did not have pneumoconiosis but rather usual interstitial pneumonia and idiopathic pulmonary fibrosis.

Finally, Dr. Perper reviewed Miner's lung biopsy slides. He diagnosed Miner with coal workers' pneumoconiosis, interstitial pulmonary fibrosis type; centrilobular emphysema, marked and honeycombing; and sclerosis of intrapulmonary blood vessels, severe, consistent with pulmonary hypertension and cor pulmonale. He based this diagnosis on Miner's pulmonary interstitial fibrosis with the presence of slight to moderate deposition of anthracotic pigment and the presence of birefringent silica crystals.

Dr. Perper was the only physician to diagnose Miner with coal workers' pneumoconiosis. I find that his opinion well-documented. However, a lung biopsy showing black pigment or anthracotic pigment does not constitute a finding of pneumoconiosis. There must be evidence that the lung tissue has reacted to the embedded coal deposits in order to qualify as a diagnosis of pneumoconiosis. Dr. Perper based his diagnosis on Miner's pulmonary interstitial fibrosis and the presence of slight to moderate anthracotic pigment. Dr. Perper fails to make any connection between the fibrosis in Miner's lungs and the existence of the anthracotic pigment. As such, I must give his opinion less weight because it is not well-reasoned. Although some of the other physicians noted black pigment in Miner's lungs, they concluded that the fibrosis and the pigmentation were unrelated. Therefore, I conclude that Claimant has failed to prove that Miner suffered from pneumoconiosis based on the biopsy evidence.

Analysis of Medical Opinions

Medical Opinion Guidance

I must next consider the medical opinions. The Claimant can establish that he suffers from pneumoconiosis by well-reasoned, well-documented medical reports. A "documented" opinion is one that sets forth the clinical findings, observations, facts, and other data upon which the physician based the diagnosis. *Fields v. Island Creek Coal Co.*, 10 B.L.R. 1-19, 1-22 (1987). An opinion may be adequately documented if it is based on items such as a physical examination, symptoms, and the patient's work and social histories. *Hoffman v. B&G Construction Co*., 8 B.L.R. 1-65, 1-66 (1985); *Hess v. Clinchfield Coal Co*., 7 B.L.R. 1-295, 1-296 (1984); *Justus v. Director, OWCP*, 6 B.L.R. 1-1127, 1-1129 (1984). A "reasoned" opinion is one in which the judge finds the underlying documentation and data adequate to support the physician's conclusions. *Fields*, above. Whether a medical report is sufficiently documented and reasoned is for the judge to decide as the finder-of-fact; an unreasoned or undocumented opinion may be given little or no weight. *Clark v. Karst-Robbins Coal Co*., 12 B.L.R. 1-149, 1-155 (1989) (en banc). An unsupported medical conclusion is not a reasoned diagnosis. *Fuller v. Gibraltar Corp*., 6 B.L.R. 1-1291, 1-1294 (1984). A physician's report may be rejected where the basis for the physician's opinion cannot be determined. *Cosaltar v. Mathies Coal Co*., 6 B.L.R. 1-1182, 1-1184 (1984). An opinion may be given little weight if it is equivocal or vague. *Griffith v. Director, OWCP*, 49 F.3d 184, 186-187 (6th Cir. 1995); *Justice v. Island Creek Coal Co*., 11 B.L.R. 1-91, 1-94 (1988); *Parsons v. Black Diamond Coal Co.*, 7 B.L.R. 1-236, 1-239 (1984).

The qualifications of the physicians are relevant in assessing the respective probative values to which their opinions are entitled. *Burns v. Director, OWCP*, 7 B.L.R. 1-597, 1-599 (1984). More weight may be accorded to the conclusions of a treating physician as he or she is more likely to be familiar with the miner's condition than a physician who examines him episodically. *Onderko v. Director, OWC*P, 14 B.L.R. 1-2, 1-6 (1989). However, a judge "is not required to accord greater weight to the opinion of a physician based solely on his status as the miner's treating physician. Rather, this is one factor which may be taken into consideration in … weighing … the medical evidence …" *Tedesco v. Director, OWCP*, 18 B.L.R. 1-103, 1-105 (1994).

Balancing Conflicting Medical Opinions

Drs. Vuskovich, Caudill, Morehead, Broudy, Sundaram, Kleinerman, Caffrey, Jarboe, and Tuteur all determined that Miner did not have pneumoconiosis. Dr. Perper diagnosed Miner with coal workers' pneumoconiosis. I find that all of these reports are well-reasoned and well-documented, because the physicians relied on objective medical data, past medical reports, and/or biopsy slides. Dr. King diagnosed Miner with chronic obstructive pulmonary disease. I give Dr. King's report lesser weight because he did not include any underlying data to support his diagnosis. The great weight of the medical opinion evidence shows that Miner did not suffer from pneumoconiosis. Therefore, I find that Claimant has failed to prove by a preponderance of the medical

opinion evidence that Miner had pneumoconiosis at the time of his death. There was no mistake in determination of fact.

Cause of Death

Even if the Claimant had shown that the Miner suffered from pneumoconiosis, she would still be required to show that his death was due to pneumoconiosis. Under section 718.205(c), the Claimant can establish death due to pneumoconiosis in any of the following circumstances: (1) where competent medical evidence establishes that the Miner's death was due to pneumoconiosis; (2) where pneumoconiosis was a substantially contributing cause or factor leading to the Miner's death or where the death was caused by complications of pneumoconiosis; or (3) where the presumption set forth at § 718.304 is applicable. The presumption at § 718.304 is not applicable to this claim. Survivors are not eligible for benefits where the Miner's death was caused by a traumatic injury or the principal cause of death was a medical condition unrelated to pneumoconiosis. 20 C.F.R. § 718.205(c)(4).

Like several other federal circuits, the United States Court of Appeals for the Sixth Circuit has interpreted "substantially contributing cause" to include a hastening of the miner's death. *Griffith v. Director, OWCP*, 49 F.3d 184, 186 (6th Cir. 1995). *See Peabody Coal Co. v. Director, OWCP*, 972 F.2d 178, 183 (7th Cir. 1992); *Shuff v. Cedar Coal Co.*, 967 F.2d 977, 980 (4th Cir. 1992). This interpretation means that any acceleration of the Miner's death that is attributable to pneumoconiosis will entitle the Claimant to benefits. *Griffith*, 49 F.3d at 186.

There are eleven medical reports in the record. Drs. Vuskovich, King, Caudill, Morehead, Kleinerman, and Caffrey did not offer opinions regarding Miner's cause of death. Drs. Broudy, Sundaram, Jarboe, Tuteur, and Perper all opined on Miner's cause of death. All of these reports are well-reasoned and well-documented as they I have already determined. Dr. Broudy found that Miner's cause of death was progressive usual interstitial pneumonia due to idiopathic pulmonary fibrosis. Dr. Sundaram opined that the complications from idiopathic pulmonary fibrosis led to Miner's death. Dr. Jarboe concluded that Miner died from respiratory failure caused by usual interstitial pneumonia/pulmonary fibrosis. Dr. Tuteur determined that Miner died as a result of progressive usually interstitial pneumonitis (idiopathic pulmonary fibrosis with inflammation). Finally, Dr. Perper claimed that Miner had pneumoconiosis and that it was a substantially contributing cause of his death. Of the five physicians who commented on Miner's cause of death, only Dr. Perper determined that his death was due to pneumoconiosis. The other physicians explained why they disagreed with Dr. Perper's conclusions. Because Claimant has failed to prove by a preponderance of the evidence that Miner's death was due to pneumoconiosis, I find no mistake in determination of fact.

Summary

In the instant case, Claimant failed to prove by a preponderance of the evidence that Miner suffered from pneumoconiosis.

FINDINGS AND CONCLUSIONS REGARDING ENTITLEMENT TO BENEFITS

The Claimant has failed to meet her burden to establish that the Miner had pneumoconiosis and that his death was caused by pneumoconiosis. I find that there was no mistake in determination of fact and that Claimant is not entitled to benefits under the Act.

ATTORNEY FEES

The award of an attorney's fee under the Act is permitted only in cases in which the Claimant is found to be entitled to benefits. See Section 28 of the Longshore and Harbor Workers' Compensation Act, 33 U.S.C. § 928, as incorporated into the Black Lung Benefits Act, 30 U.S.C. § 932. Since benefits are not awarded in this case, the Act prohibits the charging of any fee to the Claimant for services rendered to her in pursuit of this claim.

ORDER

The claim for benefits filed by the Claimant on December 1, 1997, is hereby DENIED.

A

WILLIAM S. COLWELL
Administrative Law Judge

Washington, D.C.
WSC:pah

NOTICE OF APPEAL RIGHTS

If you are dissatisfied with the administrative law judge's decision, you may file an appeal with the Benefits Review Board ("Board"). To be timely, your appeal must be filed with the Board within thirty (30) days from the date on which the administrative law judge's decision is filed with the District Director's office. *See* 20 C.F.R. §§ 725.458 and 725.459. The address of the Board is: Benefits Review Board, U.S. Department of Labor, P.O. Box 37601, Washington, D.C. 20013-7601. Your appeal is considered filed on the date it is received in the Office of the Clerk of the Board, unless the appeal is sent by mail and the Board determines that the U.S. Postal Service postmark, or other reliable evidence establishing the mailing date, may be used. *See* 20 C.F.R. §

802.207. Once an appeal is filed, all inquiries and correspondence should be directed to the Board.

After receipt of an appeal, the Board will issue a notice to all parties acknowledging receipt of the appeal and advising them as to any further action needed.

At the time you file an appeal with the Board, you must also send a copy of the appeal letter to Allen Feldman, Associate Solicitor, Black Lung and Longshore Legal Services, U.S. Department of Labor, 200 Constitution Ave., NW, Room N-2117, Washington, D.C. 20210. *See* 20 C.F.R. § 725.481.

If an appeal is not timely filed with the Board, the administrative law judge's decision becomes the final order of the Secretary of Labor pursuant to 20 C.F.R. §